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Causes of Early Human Population Growth

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ABSTRACT The archaeological record indicates large increases in human population coincident with the emergence of food production about 10,000 years ago. The cause of the growth is unclear. Extreme views attribute the change to increases in the birth rate or to decreases in the death rate. Many argue that sedentism led to improved ovarian function and higher fertility through higher caloric intakes or reduced activity levels. Similarly, shortened lactation periods may have reduced birth spacing and increased fertility. Others attribute the rise in population to decreases in mortality, arguing that the evidence from skeletal populations indicates improvements in health and the expectation of life at birth, though others use the same evidence to argue that mortality increased.

An analysis presented here draws on findings that indicate substantial increases in the survival of young children as populations switch from nomadic to sedentary lives. Projections indicate that this improvement in child survival is so critical that it may be followed by substantially larger decreases in survival at later ages, yet result in higher population growth rates and reduced expectation of life at birth. Increases in the birth rate are not necessary for population growth, even when overall mortality increases. Large increases in overall mortality can be associated with large increases in population. Because positive population growth can occur while the expectation of life at birth declines, this analysis shows that this summary statistic is not an appropriate indicator of population fitness.

Throughout the bulk of our history, humans have lived a mobile life foraging wild plant and animal foods. The switch to reliance on agriculture began about 10,000 years ago when the earliest signs of plant and animal domestication appear in the archaeological record. This shift in resource base was generally accompanied by a switch from a nomadic to a sedentary lifestyle in those groups that took up agriculture. The consequences of the shift from a mobile to a settled life may have been dramatic as the rise of food production is coincident with large increases in the human population worldwide (Cohen, 1989).

The dynamics of population during this important phase of human history are relevant to a number of issues in anthropology. Since the structure of a population corresponds to the cultural environment of its individuals, a population's demography must be known for archaeologists to fully reconstruct its culture (see Howell, 1982, for an interesting perspective on this). Understanding the demographic processes accompanying the agricultural transition is vital for correctly sequencing growth of the human population and the advent of agriculture. Presently it is unknown whether population growth is a cause or effect of plant and animal domestication. The response to the change in re-

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source base may also clarify our understanding of the life history parameters of our species. Many view variations in human reproductive and survival patterns as responses to environmental variation. To what extent the variation reflects evolved responses is disputed.

Demographic evidence relating to the transition to agriculture is scarce and difficult to interpret. Some progress has been made toward developing biological models of human mortality during the life span (Gage and Dyke, 1986; Gage, 1988, 1989, 1990; Gage and Mode, 1993) and refining techniques for inferring vital rates of past populations from skeletal material (Sattenspiel and Harpending, 1983; Johansson and Horowitz, 1986; Paine, 1989; Milner et al., 1989; Konigsberg and Frankenberg, 1994). However, mortality patterns and levels vary widely among human populations, with rates during the first 5 years of life varying perhaps most widely of all. Little is currently known about the ultimate cause of the variation.

Nevertheless, studies of modern populations indicate that early child mortality rates may decline dramatically among nomadic populations who become sedentary. One of the clearest examples comes from !Kung Bushmen of the northern Kalahari desert of Botswana. Child mortality decreased by nearly 75 percent among !Kung who became sedentary following the expansion of Herero and other cattle farmers into their range in the last half of this century (Howell, 1979; Pennington and Harpending, 1993; Harpending and Wandsnider, 1982; Lee, 1979). Increased access to milk and other high protein weaning foods at the cattleposts is most likely the cause of the improvements in child survival.

Elsewhere (Pennington, in preparation) I suggest that similar declines in early child mortality rates may characterize differences between nomadic and sedentary groups in prehistory. Below I show that these declines can have large, intuitive effects on population growth (r) and large, counterintuitive effects on the expectation of life at birth ($\mathring{e}_0$).

I begin with a discussion of problems and strategies of inferring information from paleodemographic data. Current techniques do not recognize the importance of variation in the age pattern of mortality. Paleodemographers have relied on standard life tables from industrialized populations, such as the tables compiled by Coale and Demeny (1983). Few of these take into account the mortality experiences of nonindustrialized countries that are more likely to reflect the experience of the nonmodern populations of interest to anthropologists. Inferences based on an inappropriate model life table can lead to seriously biased conclusions. Below I use a model developed by Brass (1975) and modified by Ewbank et al. (1983) to describe changes in mortality patterns that mimic the proposed improvements in child survival following the transition to agriculture. My purpose is to make sense of seemingly paradoxical population statistics as well as show that they are consistent with interpretations of the archaeological record.

MEASURING POST POPULATIONS

Stable population models

Population growth occurs when the number of births in a closed population outnumbers the deaths. Extreme views attribute the rise in population associated with agriculture to either decreases in the death rate or to increases in the birth rate, though in fact changes in both parameters are possible. Cohen (1989), for example, argues that settlement patterns associated with large populations produced large increases in the death rates so that even larger increases in the birth rate must have occurred to achieve population growth.

Stable population models have provided insight into some aspects of human population dynamics during the transition to agriculture. Under conditions of constant death rates (δ) and birth rates (γ), the growth rate r ($r = \gamma - \delta$) and the age distribution of a population will become constant and stable after only a few generations. Until relatively recently, human population groups were small, with foraging populations often numbering in bands of less than 100. Even in reasonably large populations, stochastic fluctuations in birth and death rates can cause large deviations from stability. These oscillations can pose significant problems for

anthropologists typically interested in small populations of only a few thousand individuals or less. However, these short term fluctuations become invisible in the long term. Consequently, anthropologists can use stable population theory to infer birth and death rates of past societies when the demographic information is of sufficient temporal depth.

Archaeologists concerned with reconstructing the culture of human populations in the last 10,000 years have used demographic reconstructions of prehistoric populations to understand how birth and death rates changed when populations in particular regions switched from foraging to farming (Acsádi and Nemeskéri, 1970). Paleodemographers have traditionally relied on life tables constructed from age-at-death distributions of skeletal populations to examine trends in mortality rates. If a stationary population (a stable population in which $r = 0$) can be assumed, it is possible to compute life tables from the ages-at-deaths of skeletal populations. In the special case of a stationary population, the probability of dying during an age interval ${}_aq_x$ (where x is the age beginning the interval and a is the width of the interval) can be calculated because the total size of the skeletal population reflects the size of the population at risk. The numbers of deaths at each age reflect the attrition of this population, so they provide information about those who died and survived each age.

Stationary populations have a number of convenient properties that make them especially attractive to demographers working with limited data. When $r = 0$, mean age at death (μ) is equal to $\mathring{e}_0$, and these in turn are equal to $1/\delta$ and $1/\gamma$ (Coale, 1972). In a stationary population the single statistic μ can be easily calculated from skeletal age-at-death distributions, producing an extraordinary amount of demographic information.

Unfortunately, the assumption of population stationarity is a strong one. Life tables of skeletal populations can grossly overestimate mortality rates when the life times of a skeletal population span a period of positive population growth. In this case, individuals who died early in the series would have died out of a smaller population than individuals who died more recently. While the numbers who died at each age are known, the numbers who survived are not. Even small deviations from the conditions of stationarity invalidate the special relationships among μ, $\mathring{e}_0$, γ, and δ. For example, in the stationary Coale and Demeny (1983) model West female life table, level 10, we have $\mathring{e}_0$ and μ equal to 42.50 and the per 1,000 birth and death rates equal to 23.53. In a stable population dying at the same rate but growing 0.5 percent annually, μ becomes 37.81 and the per 1,000 birth and death rates become 27.50 and 22.50. However, were we to use μ to estimate the birth and death rates in this slowly growing population, we would underestimate the true birth rate by about 4 percent and overestimate the death rate by about 15 percent.

In some cases, false assumptions of stationarity have led demographers to make absurd and circular inferences about population dynamics. For example, after finding that a population is growing, some have erroneously assigned birth rates that differ from rates using the formulas derived from stationary population models. This is algebraic nonsense, since, by definition, a population's birth and death rate must be the same if it is stationary. In the worst cases, anthropologists have estimated the death rate δ from the mean age at death μ and assigned the residual to the birth rate γ. One might just as well estimate the birth rate from the mean age at death and assign the residual to the death rate; in fact, this latter calculation happens to provide better approximations than the former (Sattenspiel and Harpending, 1983; Horowitz et al., 1988; Johansson and Horowitz, 1986; Konigsberg and Frankenberg, 1994).

In general, the special relationships among μ, $\mathring{e}_0$, and δ are not robust under conditions of nonzero population growth. Sattenspiel and Harpending (1983) demonstrated that populations with identical $\mathring{e}_0$'s but different γ's were associated with widely varying values of μ. In other words, μ is very sensitive to fluctuations in the birth rate. This principle is easy to see by viewing the Coale and Demeny (1983) regional model life tables. The tables list basic demographic sta-

tistics for 25 levels of mortality for four regions. Each table shows that increases in r correspond to decreases in μ, even though each life table holds the level of mortality constant.

On the other hand, Sattenspiel and Harpending (1983) showed that μ provides a fairly robust estimate of γ under widely varying population growth rates. Holding birth rates constant, they showed that large changes in death rates negligibly influenced the mean age at death. Horowitz et al. (1988) pointed out that the gap between μ and γ is unacceptably wide when population growth (positive or negative) is high or when the expection of life is high. However, such high growth rates and survival schedules are probably not general characteristics of prehistoric populations, so the findings of Sattenspiel and Harpending (1983) are probably valid for the variation expected from paleodemographic data. Konigsberg and Frankenberg (1994) arrived at similar conclusions. Using maximum likelihood methods, for example, they showed that the birth rate is much more reliably estimated than the death rate at the Libben site.

This means that age-at-death distributions are informative about a population's birth rate, but not its death rate, in stable, growing populations. Consequently, a great deal of information about population dynamics in prehistoric populations can still be gleaned from the single statistic μ. For example, Sattenspiel and Harpending (1983) examined μ in a sample of eastern European and northern African populations undergoing the transition to agriculture. μ was higher in the Neolithic than the Paleolithic populations, indicative of declining birth rates, suggesting that the cause of population growth in those regions was decreasing death rates.

Since the distribution of deaths in a population depends on the pattern and level of mortality and fertility, constructing a life table for a growing population from the age-at-death distribution poses a number of difficulties. One approach has been to fit the age-at-death distributions to model life tables under conditions of varying population growth (Paine, 1989). However, very large samples are needed to discriminate statistically among the many combinations of death and birth rates, and some knowledge of the age pattern of mortality is needed to choose an appropriate model life table. Howell's (1976) uniformitarian position encouraged anthropologists to adopt the Coale and Demeny (1983) model West series of life tables as their standard. Demographers now recognize that the mortality experience of many modern human populations are not captured in the Coale and Demeny tables. As I show below, the choice of the model life table impacts substantively the inferences drawn from the data.

The age-pattern of mortality

The model life tables for anthropological populations developed by Weiss (1973) are an alternative to the Coale and Demeny tables but may prove less reliable because of the small number of populations upon which they are based. Unfortunately, detailed and reliable demographic data on the mortality experiences of small, non-Western populations are still scarce. Even with reasonably large samples, it is hard to rule out stochastic error as the source of differences in mortality levels between populations. Deaths become increasingly rare as children age in all human populations, and estimates of mortality rates after early childhood become increasingly unreliable. By old age, mortality rates are once again high. However, the number of people who survive to old age is relatively small. As a result, estimates of mortality at the oldest ages tend to be based on the experiences of only a handful of people. Further error is introduced through uncertainty in determination of age among individuals in the type of populations upon which anthropologists tend to focus.

Nevertheless, reliable information about the survival of infants and young children exists for a number of populations. Table 1 lists these rates for 13 groups (from Pennington, in preparation). Rates during different periods or locations for seven of the groups are included for comparison.

The most striking feature of the data is that infant mortality (${}_1q_0$, the life table probability of dying from birth to age 1) varies drastically among these groups, and that early childhood rates (${}_4q_1$, the life table prob-

TABLE 1. Infant and child mortality rates in traditional societies

Region	Population	Mobility	Subsistence	${}_1q_0$	${}_4q_1$	Source
Africa						
Mali	Fulani (Seno-Mango)	semi-nomadic	agro-pastoralism	0.15	0.20	1
Mali	Fulani (Delta)	sedentary	agro-pastoralism	0.22	0.36	1
Botswana	!Kung	nomadic	foraging	0.22	0.22	2, 3
Botswana	!Kung	sedentary	foraging	0.18	0.06	2, 3
Gambia	Mandinka & Jola (Keneba)	sedentary	horticulture	0.24	0.34	4
Gambia	Mandinka & Jola (Manduar)	sedentary	horticulture	0.13	0.23	4
Mali	Bambara	sedentary	agriculture	0.20	0.22	1
Mali	Tamasheq (Delta)	nomadic	pastoralism	0.13	0.19	1
Mali	Tamasheq (Gourma)	nomadic	pastoralism	0.15	0.20	1
Tanzania	Datoga	semi-nomadic	pastoralism	0.21	0.10	5, 6
Kenya	Kipsigis (1945–90)	sedentary	pastoralism	0.09	0.06	7
Kenya	Kipsigis (1918–53)	sedentary	pastoralism	0.08	0.11	7, 8
Botswana	Herero (1975–86)	sedentary	pastoralism	0.06	0.03	3
Botswana	Herero (1960–74)	sedentary	pastoralism	0.12	0.06	3
Botswana	Herero (before 1959)	sedentary	pastoralism	0.13	0.09	3
Asia						
Nepal	Nyinba	sedentary	agriculture	0.22	0.14	9
Nepal	Tamang	sedentary	agro-pastoralism	0.20	0.08	10
Malaysia	Semai Senoi	sedentary	horticulture	0.22	0.12	11
N. America						
Yukon	Kutchin	nomadic	foraging	0.17	0.12	12
Yukon	Kutchin	sedentary	foragers	0.09	0.07	12
S. America						
Brazil	Yanomama (Mucajai)	sedentary	horticulture	0.14	0.09	13

Sources: (1) Hill et al., 1983, (2) Howell, 1979, (3) Pennington and Harpending, 1993, (4) Billewicz and McGregor, 1981, (5) Borgerhoff Mulder, 1991, (6) Borgerhoff Mulder, 1992, (7) Borgerhoff Mulder, personal communication, (8) Borgerhoff Mulder, 1988, (9) Levine, 1988, (10) Fricke, 1986, (11) Fix, 1977, (12) Roth, 1981, (13) Early and Peters, 1990.

ability a 1-year-old dies before turning 5) vary even more drastically. Infant mortality ranges from a low of 0.06 among recently born Herero of Botswana to a high of 0.24 among an ethnically mixed population of Mandinka and Jola in the Gambia. The lowest early childhood mortality rates are found again among the most recently born cohort of Herero children, 0.03 of whom die from age 1 to 5, while the highest rates are found among Delta Fulani in Mali, where 0.36 of children reaching the age of one die before they turn 5.

The mortality of children also varies widely within single ethnic groups. Early mortality among the children of nomadic !Kung San foragers was 0.22 but declined to 0.06 in later years when they became more sedentary and dependent on neighboring Herero pastoralists. The decline in mortality appears to be the result of increased caloric intakes, particularly from milk from domesticated animals, associated with sedentary life on Herero cattleposts (Harpending and Wandsnider, 1982; Pennington and Harpending, 1993). Sedentary !Kung children, who had more food available to them than nomadic children, were able to accumulate larger fat stores than their nomadic counterparts. These fat stores provided nutritional buffers against periodic dietary deficiencies and illness. Since Herero survival rates changed only moderately during the same period, it is unlikely that other environmental factors are responsible for the large decline in !Kung early childhood mortality (Pennington and Harpending, 1993).

Like the !Kung, the Kutchin also experienced large declines in mortality when they became sedentary. The cause is less clear. The declines in mortality associated with the transition to sedentary life in the !Kung and Kutchin, however, clearly contradict the assumption that mortality increases unilaterally in sedentary populations.

Access to cow's milk and other high protein weaning foods may explain much of the variation in child mortality rates in the table. Indeed, the lowest mortality rates are found among the cattle-keeping peoples in Table 1, but the picture is far from clear. Within narrow geographic regions and single ethnic groups, rates vary tremendously. For example, nomadic Seno-Mango Fulani children survive better than sedentary Fulani living in the Delta, and the Mandinka and Jola

living in Manduar survive significantly better than Mandinka and Jola living only a few kilometers away in Keneba. Hill et al. (1983) attribute the difference between and among the groups in their study to crowd diseases and differential access to milk, but no apparent differences in diet or settlement practices were evident between the Mandinka-Jola populations (Billewicz and McGregor, 1981).

The sample in the table, overrepresentative of African peoples, also illustrates the influence of regional factors on survival. Demographers have long recognized that West Africans experience characteristically high mortality between ages 1 and 5 (Brass et al., 1968). The typical West African pattern of child mortality is one in which ${}_4q_1$ equals or exceeds ${}_1q_0$. The West African pattern also captures the pattern found among the nomadic !Kung foragers during the first few years of life.

To what extent the West African pattern of mortality characterizes prehistoric and modern foraging populations in general is unclear. It is clear, however, that early child mortality rates are exquisitely sensitive to environmental factors. In the worst conditions, nearly half of all children die by the time they reach age 5. After infancy, the most dangerous period of an African child's life occurs at weaning (c.f. Rosetta and O'Quigley, 1990; Garenne, 1981), suggesting that subsistence strategies may be keyed to parental strategies aimed at improving the survival of children.

Health of past populations

Information about the health of prehistoric peoples has both supplemented and confused efforts to understand paleodemographic information. Paleopathologists identify sources of poor health in prehistoric populations from the signatures diseases leave on skeletal remains. That indicators of nutritional status, such as enamel hypoplasias, porotic hyperostoses, and Harris lines, are generally more frequent in sedentary than mobile peoples is hardly disputed, but what the differential pattern of disease means is.

At first glance, increasing frequencies of skeletal pathologies in populations undergoing transition from nomadic to sedentary life would seem to suggest declining health. Declining health suggests increasing mortality. Well-known principles of epidemiology are consistent with this interpretation. Infections spread more readily in sedentary populations, since typically they have larger pools of susceptible hosts to maintain diseases than in nomadic groups. Human waste cultivates disease and accumulates more rapidly in sedentary groups, and those with inadequate sewage removal live in proximity to this disease-harboring waste. Water supplies are easily contaminated in these conditions, facilitating the spread of water-borne pathogens such as typhoid as well as diarrheal diseases. Agricultural populations become susceptible to the diseases of their livestock either by becoming incidental hosts to zoonotic diseases or by contracting mutated forms of animal pathogens. Many diseases that have decimated human groups historically, such as tuberculosis and influenza, may have their pathological origins among domestic livestock (Burnet and White, 1972). These hazards of sedentary life must be balanced against comparable disadvantages of nomadic life in which people may need to acquire immunity to new pathogens everytime they move.

Because many stresses only leave evidence on victims who survive a disease or bout of nutritional stress, some anthropologists suggest that indicators of poor health left on skeletons may actually indicate better health and survival (Wood et al., 1992). For example, a paleopathologist examining the skeletons of children who died from their first episode of nutritional stress will find no signs of nutritional stress on the skeletons. In contrast, children who experienced the same episode of nutritional stress but survived because they were in other ways healthier may have their skeletons marked by the event. As a result, the paleopathologist would find signatures of nutritional stress on the healthy children but none in the children who died.

This problem of interpretation is in some ways the inverse of the difficulties posed in constructing life tables of populations undergoing growth. In this case, the paleopathologist knows how many individuals survived a disease (or at least survived long enough

for the disease to leave a mark) but does not know how many individuals died from the disease.

Several other related issues confound clear interpretation of paleopathological data. Only a handful of diseases leave distinguishing marks on the human skeleton, and pathogens afflicting past peoples may no longer be a known source of disease. Consequently, anthropologists reconstructing the health and population of prehistoric peoples may be missing essential information about the past environment. Interactions among the many competing risks add another plane of complexity. All causes of death or illness compete, which means that the prevelance of one risk influences the prevalence of others. The emerging pattern of disease under ecological or environmental change is not easy to predict. One of the more interesting examples of this principle is the relationships among the treponemal diseases. In the 1950s the World Health Organization (WHO) undertook a campaign to eradicate yaws. As part of this effort, they injected millions of people with massive doses of penicillin. Although yaws yet remains to be eradicated, the WHO did succeed in reducing the incidence of this disease (Guthe et al., 1972). The effort, however, resulted in the rise of the more serious treponal diseases. People who were saved from yaws as children did not develop immunity to treponema and, thus, were more likely to contract venereal syphilis as adults. Similar interactions among competing causes have been well documented (c.f. Manton and Stallard, 1984) and have certainly influenced the pattern of mortality during the evolution of human societies.

Fertility

Despite these difficulties, many believe that the weight of evidence indicates that human mortality rates increased when populations began settling down. Consequently, they argue that increases in fertility associated with farming must be responsible for the increase in population (Cohen, 1989). Several mechanisms have been proposed to understand how the switch to farming may have caused fertility to increase. For example, higher caloric intakes or reduced activity levels associated with sedentism may result in improved ovarian functioning, and, ultimately higher female fertility (c.f. Bentley, 1985; Ellison et al., 1989; Ellison, 1990). Similarly, shortened lactation periods resulting from sedentism may reduce birth spacing and increase fertility. However, comparisons of the fertility of nomadic foraging populations with the fertility of sedentary agriculturalists do not show a consistent difference in their fertility levels (Campbell and Wood, 1988; Bentley et al., 1993).

On the other hand, there is general agreement that the substantial variation in reproductive rates cannot be explained by subsistence system alone. Average levels of fertility among populations practicing different modes of subsistence vary so widely that their ranges overlap extensively (Wood, 1994). This means that a population's subsistence mode is not a strong predictor of its fertility, since virtually all fertility rates are found with similar frequencies in all types of noncontracepting populations. For example, given information that a population has a total fertility rate (TFR) of, say, 5.0, one cannot confidently conclude that the population lives by foraging, horticulture, or intensive agriculture.

Neither the study by Campbell and Wood (1988) nor that by Bentley and colleagues (1993) used fertility rates from all the demographic literature they examined to arrive at their conclusions. Rather, each excluded some populations in their analysis based on criteria relevant to the purpose of the study. Many of the criteria, such as poor data quality, could be justified in any study of fertility. Other criteria, such as excluding populations with depressed fertility due to factors such as disease, limit the generality of their findings to other contexts. Fertility-inhibiting diseases have a long history in human populations (Pennington and Harpending, 1993; McFalls and McFalls, 1984), and one might well postulate that the magnitude of the effects of these diseases on fertility is linked to the subsistence system of populations. Since infectious diseases are more easily spread in dense, sedentary populations, those diseases that impact fertility should be more prevalent in them as well.

Understanding the relationships among

disease, health, and fertility in prehistory introduces difficulties akin to understanding the relationships among disease, health, and survival in skeletal populations described above. For example, diseases such as syphilis and tuberculosis have some impact on fertility (McFalls and McFalls, 1984; Pennington and Harpending, 1993) and leave their signatures on skeletons. However, diseases that we know severely impact fertility in modern populations are undetectable to the paleopathologist. Pelvic inflammatory disease (PID), caused by gonorrhea, chlamydia, and other bacteria, is the probable cause of dramatically low fertility rates in much of central Africa (Frank, 1983; Caldwell and Caldwell, 1983) today. Certainly these diseases and others affected the fertility of prehistoric populations, although the most virulent of them may have had little effect on small dispersed groups of humans. To understand the relationship between sedentism and fertility, anthropologists need to understand the evolutionary role of disease on fertility as well (Pennington and Harpending, 1991). In this sense, disregarding impaired fertility populations in the study of reproduction is like studying only disease-free populations in the study of survival.

Modeling population growth

Given these difficulties, arriving at any conclusions about the demography of past peoples seems impossible. The postulated increases in mortality rates must be offset by similar increases in the birth rate for positive population growth to occur, but I argued above against evidence for changes in birth rates. This means that increases in population must have occurred under conditions of increasing mortality and stable fertility. At first glance, this assertion seems hopelessly paradoxical. But most studies relating mortality levels to subsistence system have focused on average levels of mortality rates across the life span. Below I show that minor modifications in the age pattern of mortality can produce sizable increases in population, even if fertility rates stay constant. The modifications are consistent with the archaeological evidence indicating rises in overall mortality and with data from the !Kung and other anthropological populations indicating increases in child survival associated with sedentism.

METHODS

Below I generate a series of hypothetical but plausible life tables to model changes in the level and pattern of mortality during the transition to agriculture. The life tables are produced using a parametric model (the Brass relational, modified by Ewbank et al., 1983) that generates new life tables from a baseline standard. The parameters of the model modify the basic shape of the standard life table to produce new life tables with different but related patterns of survival.

Next, I produce a series of life tables to simulate changes in mortality during the agricultural transition. This series of life tables produces higher overall levels of mortality, consistent with the findings of Cohen (1989) and others who believe that mortality increased during the transition. Mortality rates during early childhood, however, are lower than in the standard, consistent with findings described above and documented elsewhere (Pennington, in preparation). The expectation of life at birth ($\mathring{e}_0$) in the new relational tables is lower than in the standard, reflecting the overall higher levels of mortality.

Finally, the life tables are projected to estimate the intrinsic growth rate r associated with each life table corresponding to an arbitrary schedule of fertility. The growth rate is used to estimate other relevant demographic parameters, and their correspondence to the $\mathring{e}_0$ of the relational tables is discussed.

Relational life tables

The Brass relational model. The relational life table system developed by Brass (1975) is a system in which a given survival schedule can be used to produce any number of related life tables. Specifically, the Brass system derives one survival schedule from another using the logit transformation of the life table probabilities of survival (l_x) from birth to each age x. The model is,

$$\text{logit}(1 - l_x) = \alpha + \beta\text{logit}(1 - l_x^*),$$

where l_x^* is the standard life table and l_x is

the life table generated. The logit is defined as

$$\text{logit}(1 - l_x) = 0.5 \ln\left(\frac{1 - l_x}{l_x}\right).$$

This system adequately fits a wide variety of empirical life tables (Carrier and Hobcraft, 1971). At the extreme ends of the life span, however, the fit of the model is often unsatisfactory. Modifications to the Brass system (e.g., Zaba, 1979) have broadened the range of life tables that can be adequately fit by the relational system.

The four-parameter logit system. Ewbank et al. (1983) proposed a logit life table system with two additional parameters. Their four-parameter life table system improved the flexibility of the Brass model so that virtually any pair of life tables can be fit. This model is

$$\text{logit}(1 - l_x) = \alpha + \beta T(l_x^*; \kappa, \lambda),$$

where

$$T(l_x^*; \kappa, \lambda) = \begin{cases} \dfrac{\left(\dfrac{1 - l_x^*}{l_x^*}\right)^{\kappa} - 1}{2\kappa} & \text{for } l_x^* \geq 0.5 \\ \dfrac{\left(\dfrac{1 - l_x^*}{l_x^*}\right)^{-\lambda} - 1}{-2\lambda} & \text{for } l_x^* < 0.5 \end{cases}$$

The new (relational) survival rates are obtained by solving for l_x:

$$l_x = \frac{1}{\exp[2\alpha + 2\beta T(l_x^*; \kappa, \lambda)] + 1}.$$

Effects of the parameters. As κ and λ approach 0, the four-parameter model approaches the two-parameter Brass model. The effects of increases and decreases in the four parameters on survival curves are shown in Figure 1. The interpretation of them is as follows.

α alters the level of mortality in the relational table. When α is greater than zero, the level of mortality in the new relational table will be higher than it was in the standard life table. When α is less than 0, the new relational table will have a lower overall level of mortality. Different values of α change the overall level of mortality between life tables but do not change the age pattern of mortality. Incremental increases in α in the models below simulate hypothesized increases in mortality during the Neolithic.

β alters the 'slope' of mortality. Deviations from 1.0 cause the survival curve to pivot at the point at which $l_x = 0.5$. When β is greater than 1, the survival curves pivots clockwise. As a result, childhood mortality is relatively lower in the relational table than in the standard table but higher at older ages. A value of β between 0 and 1 pivots the life table counterclockwise, producing a relational life table in which early mortality is relatively higher than later mortality. This parameter was used to modify the age pattern of mortality in the simulation below.

κ changes the steepness of mortality at the earliest ages but does not affect mortality at the oldest ages. When κ is greater than 0, survival declines more rapidly at young ages in the relational table than in the standard. When κ is less than 0, survival at young ages declines more slowly in the relational table than in the original standard. This parameter was used to fine-tune the fit of the relational life tables during early childhood to the rates described below.

λ changes the steepness of mortality at the oldest ages. When λ is greater than 0, survival at later ages will decline more slowly in the relational tables than in the standard. When λ is less than 0, survival at later ages will decline more rapidly than in the standard. This parameter is ignored in the models below.

The choice of the standard. My choice of the baseline (standard) life table from which I generated the relational life tables is somewhat arbitrary. I chose the Brass African standard (Carrier and Hobcraft, 1971) because early childhood mortality is higher in

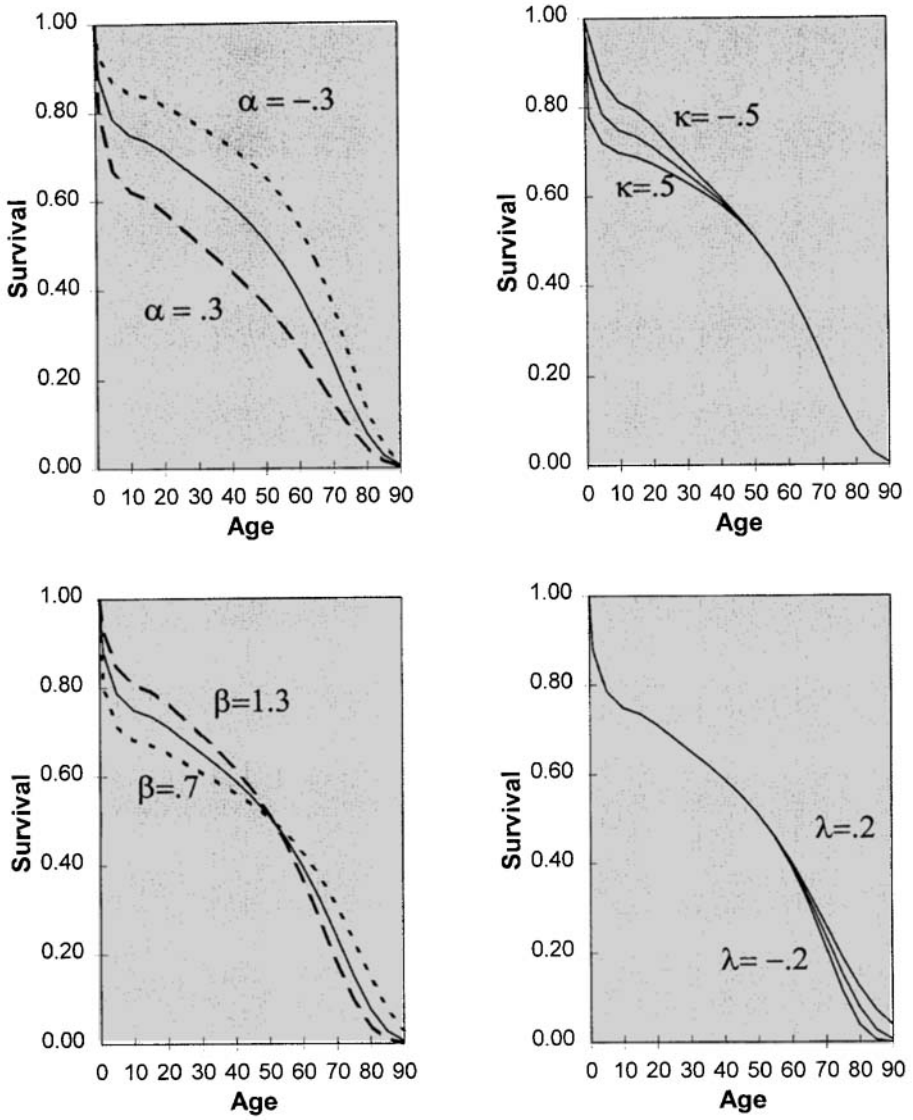

Fig. 1. Effects of the parameters in the modified relational life table system. The African standard is the baseline model, and the figures show how increases and decreases in each parameter increase and decrease survival throughout the life span.

this life table than in models of other modern populations. In this life table, ${}_1q_0$ and ${}_4q_1$ are approximately equal, mimicing the age-pattern of mortality among nomadic !Kung during the first 5 years of life. Since my purpose is to model the effects of critical changes in the age pattern of mortality on population growth, any life table with this pattern would suffice.

I increased overall levels of mortality by incrementally increasing α. I chose values of β and κ so that ${}_1q_0$ was held constant in all of the tables while ${}_4q_1$ was decreased by 50 percent from the standard in each relational table. In other words, a newborn's chances of dying in its 1st year of life is identical in the standard and in each of the relational tables. A child of exactly age 1, however, has half the chance of dying before it reaches age 5 in each of the relational tables compared to

the standard. This pattern models apparent changes in survival among the modern anthropological populations described in Table 1, particularly the former !Kung foragers of Botswana.

For convenience, infant mortality was held constant in all of the tables. In reality, mortality risks probably change continuously in that declines in infant mortality probably coincide with declines in early childhood mortality. On the other hand, if exploitation of dairy products and other high quality weaning foods associated with plant and animal domestication is part of parental reproductive strategies, then their largest effects will occur during early childhood when weaning typically occurs. Detailed demographic studies in those populations with the highest early childhood mortality rates indicate sharp rises around weaning (e.g., Garenne, 1981). Few anthropologists have data of this quality, and so for clarity of presentation only early childhood mortality was modified in the relational tables.

Mortality beyond age 5 increases gradually between the relational tables. To what extent the generated life tables mimic patterns of mortality in known populations is discussed below.

Population projection

To assess the importance of variation in patterns of survival on population growth, I projected the life tables using standard matrix projection methods (Keyfitz, 1985) and the formulas described by Pennington and Harpending (1993). Given a schedule of mortality and fertility and a starting population, population projection estimates the number of individuals at each age in the future. The properties of the Leslie matrix (Leslie, 1945, 1948) used in projections are well known. The basic form of this matrix (for the female population) is

$$A = \begin{bmatrix} F_1 & F_2 & F_3 & \cdots & F_j \\ P_1 & 0 & 0 & \cdots & 0 \\ 0 & P_2 & 0 & \cdots & 0 \\ \vdots & \ddots & \ddots & \cdots & \vdots \\ 0 & 0 & \cdots & P_{j-1} & 0 \end{bmatrix}.$$

The diagnoal entries P_i, $i = 1,2,3, \ldots, j$, are the probabilities that females in age class i survive from time t to $t + 1$. The F_i entries in the top row are the expected numbers of births to females in the ith age classes from time t to $t + 1$. I chose a population with an intermediate fertility schedule (the age-specific fertility rates from the Herero of Botswana during 1967–76 given by Pennington and Harpending, 1993), summarized by a TFR of 5.4, for the projection. I assumed a secondary sex ratio of 0.5.

A population is projected by multiplying the $j \times j$ matrix A by a $j \times 1$ vector of population. Each $n_i(t)$ in the vector

$$n'(t) = [n_1(t), n_2(t), n_3(t), \ldots, n_j(t)]$$

is the number of females in age class i at time t. Term $n'(t)$ is the transpose of the column vector $n(t)$. For each life table, I estimated future population by iterating the process $n(t + 1) = An(t)$ from an arbitrary starting population. The intrinsic growth rate r associated with each life table was estimated when the population reached a stable age distribution. All calculations were computed using a Microsoft Excel 5.0 spreadsheet.

The choice of the schedule of age-specific fertility rates is, like the choice of the standard life table, somewhat arbitrary. Levels of marital fertility vary among human natural fertility populations, but the age-pattern does not (Wood, 1989). Herero reproductive patterns are no exception. It is unlikely that Herero fertility levels typify any modern or prehistoric population, since fertility levels vary so widely in all categories of human populations (see above). A higher (or lower) fertility schedule would produce a higher (or lower) intrinsic growth rate, but the magnitude of the differences between the life tables would not be significantly affected. In other words, inferences based on the results of the projections are not affected by the choice of the fertility schedule.

RESULTS

Four relational life tables were generated from the African standard. The parameter values used to generate them are in Table

TABLE 2. Parameter values used to generate the relational life tables and the resulting expectation of life at birth ($\mathring{e}_0$) for each table

	Standard	Relational			
α	0.0	0.0	0.3	0.6	0.9
β	0.0	1.98	3.09	4.46	5.76
κ	0.0	0.79	1.04	1.29	1.43
$\mathring{e}_0$	44	43	38	35	33

α was increased incrementally to model increases in overall levels of mortality, relative to the standard. β and κ were then chosen so that infant mortality was unchanged and early childhood mortality was reduced by one-half in the relational tables.

2. Plots of the life tables and their corresponding age-specific mortality rates are in Figure 2. The results of the projection are in Table 3.

The first relational table in Table 2 shows the effect of an initial increase in child survival rates. Overall mortality rates are not increased ($\alpha = 0.0$), but the pattern of mortality is shifted so that children survive better while adults survive worse. The expectation of life at birth for both of these tables is nearly the same, although the pattern of mortality between the tables is very different. $\mathring{e}_0$ decreases by about 10 years from the first to last relational table.[1]

From Figure 2 (right side) it is easy to see that the chances of dying at every age from birth to about 30 are higher in the standard compared to the first relational table. After this age, mortality is higher in the relational table. In the remaining three q_x curves, mortality at every age greater than 5 is higher in the relational tables compared to the standard.

Figure 2 (left side) shows that a newborn's chances of surviving to every age through 50 are lower in the standard table compared to the first relational table. On the other hand, a newborn has a better chance of surviving to older ages in the standard table. For example, a newborn surviving according to the standard life table has about a 40 percent chance of reaching age 60, while in the first relational table the chances of reaching age 60 are only about 30 percent. The chances of reaching older ages diminish rapidly as α increases in each of the relational tables. In the last relational table ($\alpha = 0.9$), less than 5 percent of newborns would survive to age 60.

The overall decline in survival throughout the life table is captured by the average number of years lived by newborns ($\mathring{e}_0$). This statistic declines by 10 years from the first to last relational table. Remarkably, the highest value of $\mathring{e}_0$ does not correspond to the life table with the highest growth rate. Table 3 shows that the first three relational tables produce populations, according to the projection, that are growing at faster annual rates (r) than the population described by the standard. $\mathring{e}_0$ is 44 years in the standard and 35 in the third relational table ($\alpha = 0.6$), but the projected relational population grows 0.1 percent faster per year than the longer-lived population. The last two columns of the table show the effect of the differences in r on population. Doubling time, the number of years required for a population growing at the rate r to double in size, is reduced by several years in three of the relational tables. The projected population is the number of descendants that would be produced by a population of 1,000 growing at the rate r after 200 years.

The standard and first relational life tables have similar $\mathring{e}_0$'s, but the projections estimate that the relational table will produce more than two times the population of the standard in only 200 years. Expectation of life at birth in the last relational table is 11 years lower than in the standard, yet the projections produce populations growing at identical rates. Although mortality is higher in the relational life table after age 5, higher survival during the critical weaning period means that a larger proportion of newborns are surviving to reproduce.

DISCUSSION

Demographic techniques provide anthropologists with the potential to infer the causes of shifts in subsistence strategies from the sequence of demographic change surrounding these shifts. However, the limitations of paleodemographic data, such as

[1] $\mathring{e}_0$ is defined as

$$\sum_{x=0}^{\omega} \frac{(l_x + l_{(x+a)}) \cdot a}{2},$$

where a is the width of each age interval and ω is the end of the life span.

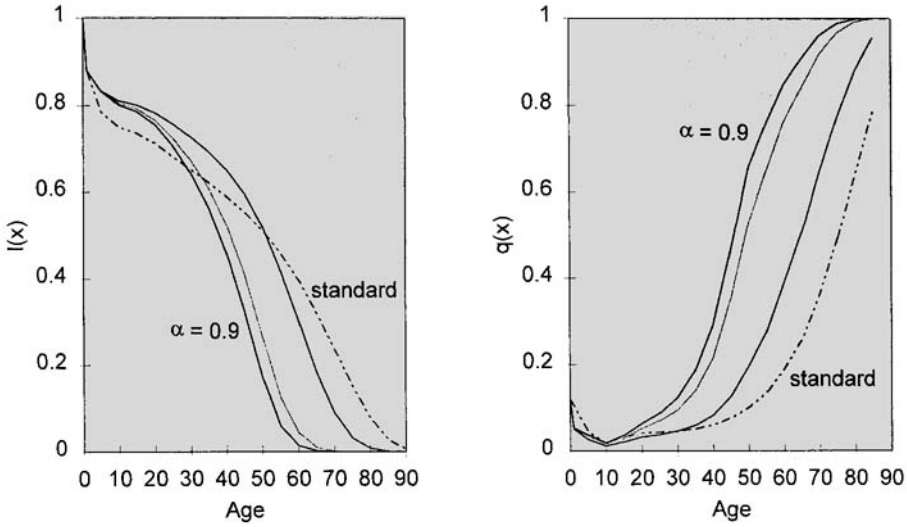

Fig. 2. Survival (l_x) and mortality (q_x) schedules in the African standard and relational life tables.

TABLE 3. Effects of changes in mortality on the expectation of life at birth ($\mathring{e}_0$), annual intrinsic growth (r) *rate, doubling time, and population size*

Life table	$\mathring{e}_0$	r	Doubling time	Projected population
African standard	44	0.0220	32	81,500
Related table				
α = 0.0	43	0.0258	27	181,000
α = 0.3	38	0.0244	28	122,000
α = 0.6	35	0.0233	30	99,500
α = 0.9	33	0.0219	32	81,500

The projected population is the number of descendants a population of 1,000 growing at the rate r for 200 years would produce. Infant mortality is held constant and early childhood mortality is decreased from the standard by one-half in the relational tables. Increases in α correspond to increases in overall mortality.

the use of skeletal age-at-death information in life table construction, hinders reconstructing the population dynamics of prehistoric peoples. This paper has emphasized the central role of changing mortality patterns on the growth of populations during the agricultural revolution. In particular, I have suggested that trade-offs between the survival of parents and their offspring may cause large increases in population. Ratios of juvenile and adult mortality have long been recognized as important life history variables (Horn and Rubenstein, 1984), but Darwinian principles are infrequently used to understand the costs and benefits of human innovation in prehistory.

In this paper I have described evidence for declining mortality among the children of nomadic peoples who become sedentary. My analysis shows that this decrease is so critical that levels of mortality can increase drastically at later ages and still produce an increasing population. The decline in early childhood mortality modelled here was 50 percent, but may be even greater in real populations. For example, the mortality of !Kung children declined by more than two-thirds when they became sedentary (see Table 1 and Harpending and Wandsnider, 1982, Howell, 1979, and Pennington and Harpending, 1992).

Also remarkable is the effect of changes in the age-pattern of mortality on the age-at-death distribution.[2] Table 4 shows the ex-

[2]If D_x is the expected number of deaths in each age class x, then the proportion d_x of deaths in each age class is

$$\frac{D_x}{\sum_{x=0}^{\omega} D_x},$$

with

$$D_x = \frac{l_x \cdot q_x}{e^{r\frac{1}{2}(x+x+a)}}.$$

ω is the end of the life span, a is the width of the age interval, and e is the base of the natural logarithm. In the stationary population, the denominator reduces to 1.

TABLE 4. Expected age-at-death distribution (d_x) of projected life tables

	Life table				
Age	Standard	$\alpha = 0.0$	$\alpha = 0.3$	$\alpha = 0.6$	$\alpha = 0.9$
0	0.251	0.295	0.256	0.239	0.224
1	0.186	0.106	0.097	0.087	0.085
5	0.065	0.046	0.048	0.048	0.050
10	0.023	0.018	0.019	0.020	0.022
15	0.033	0.028	0.032	0.034	0.038
20	0.039	0.036	0.044	0.050	0.057
25	0.035	0.036	0.046	0.055	0.065
30	0.031	0.036	0.049	0.062	0.074
35	0.030	0.040	0.056	0.073	0.087
40	0.030	0.045	0.066	0.086	0.099
45	0.032	0.055	0.079	0.098	0.100
50	0.035	0.065	0.084	0.085	0.068
55	0.037	0.064	0.064	0.044	0.024
60	0.040	0.058	0.038	0.016	0.006
65	0.040	0.040	0.016	0.004	0.001
70	0.037	0.022	0.005	0.001	0.000
75	0.029	0.008	0.001	0.000	0.000
80	0.017	0.002	0.000	0.000	0.000
85	0.007	0.000	0.000	0.000	0.000
90	0.001	0.000	0.000	0.000	0.000
95	0.000	0.000	0.000	0.000	0.000
Total	1.000	1.000	1.000	1.000	1.000

pected proportions of deaths at each age for each life table growing at the rate projected in Table 3. The proportion of deaths at young ages is higher in the African standard than in the relational tables. However, the proportion of deaths at the oldest ages diminishes between the relational tables as increasingly more deaths occur during middle age. The differences in the age-at-death distributions mean that a paleodemographer working with 1,000 skeletons should expect to find 208 individuals who died after age 50 if he assumes a pattern of mortality like the standard but only 31 skeletons if he assumes a pattern of mortality like the last relational table, although both projected populations are growing at about the same rate. Projection of the first relational table produces a more rapidly growing population than the standard yet the paleodemographer should expect to find about the same number individuals who died after age 50.

Examination of the age-at-death distributions above shows that variations in age-patterns of mortality influence interpretations of paleodemographic data. Some authors have suggested fitting model life tables to skeletal age-at-death distributions (Paine, 1989; Howell, 1976), but this method can produce misleading results if a table with an inappropriate age-pattern of mortality is chosen. The life tables projected in this paper clearly illustrate that small changes in early childhood mortality are enough to account for very large changes in population growth. As Table 3 shows, populations can grow substantially in just a few generations, even under conditions of unchanging birth rates and declining adult survival.

The life tables generated here do not resemble the model life tables described by Coale and Demeny (1983), but neither do most life tables constructed from paleodemographic data (Howell, 1982). However, differences between the African standard and the generated relational tables modelled herein show changes in mortality rates during early life that are consistent with patterns found in modern anthropological populations. The associated age-at-death distributions are consistent with distributions found in skeletal samples (c.f. Acsádi and Nemeskéri, 1970; Henneberg and Steyn, 1994). The finding that different patterns of mortality substantively impact population dynamics emphasizes the need for the development of extensive model life tables based on nonindustrial populations.

This paper also emphasizes the importance of parental care on child survival (Harpending et al., 1990; Pennington and Harpending, 1988). Clearly, parents who keep their children alive a little better, even at higher survival cost to themselves, may have higher reproductive success. Since r measures the mean rate at which individuals in a population replace themselves, it can be used as a measure of reproductive success. From a simple Darwinian point of view, individuals adopting the reproductive strategy that produces the highest r in a given environment will increase their genetic representation in future generations faster, compared to individuals practicing alternative strategies. The costs and benefits associated with changes in subsistence strategies must encompass the wide range of life history variables that may be influenced by local ecological factors. The Darwinian framework may provide insight into the complexities of modern and prehistoric population dynamics.

CONCLUSIONS

A population projection simulating hypothesized changes in mortality in prehistory are counterintuitive. Overall mortality levels were increased, yet projections produced populations characterized by significantly higher growth rates. The results show that increased birth rates are not required for population growth, even when overall mortality increases. The projection shows that moderate increases in child survival rates have more substantial effects on population growth than mortality at later ages. Improvements in child survival may be so critical that decreases in the expectation of life at birth of more than a decade can occur and still produce a population characterized by the same r.

The projection also shows that $\mathring{e}_0$ is not an appropriate summary statistic for comparing levels of mortality between populations with different age patterns of mortality. In the projections described here, populations with lower values of $\mathring{e}_0$ produced faster growing populations than other populations with higher values of $\mathring{e}_0$.

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